Management of Pancreatic Ascites

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G&H How does pancreatic ascites manifest in the body?

RK Pancreatic ascites is one manifestation of a pancreatic disruption. Disruptions are divided into internal or external fistulas and have a number of consequences. Internal fistulas include pseudocysts, enteric or biliary fistulas that occur in the setting of pancreatic necrosis, where the pancreatic digestive enzymes digest their way into whatever tissue happens to be contiguous to them, as well as high amylase pleural effusions and ascites. Ascites simply denotes a leak of pancreatic juice into the peritoneal cavity, whereas a pseudocyst occurs when the body walls off that pancreatic leak. The consequences of the pseudocyst depend on where the leak is and how rapidly the leak occurs. Approximately 50% of patients who develop pancreatic ascites have a concomitant pseudocyst that is leaking.

G&H What are the causes of pancreatic pseudocysts and ascites?

RK Approximately 95% of cases of pancreatic ascites are associated with chronic pancreatitis. The leak manifests upstream of a stricture or stone, and the point of least resistance for the pancreatic juice to flow is into the belly cavity rather than the duodenum, where it belongs. There are several reported cases of posttraumatic pancreatic ascites, where the tail of the pancreas has been sheared off after a motor vehicle accident, bicycle handlebar injury, or football helmet injury. Ascites can occur as a result of knife or gun wounds. It can occasionally occur after surgery for splenectomy or left nephrectomy, where the tail of the pancreas is damaged and the duct leaks juice into the belly cavity.

G&H How do patients with pancreatic ascites generally present in terms of symptoms?

RK These patients present with increased abdominal girth, weight loss, variable abdominal pain, and possibly shortness of breath if the fluid compresses the diaphragm or enters transdiaphragmatic channels resulting in concomitant high amylase pleural effusions. They may also lose proteins in their abdominal cavity from the ascites and may develop ankle swelling or anasarca.

G&H Does pancreatic ascites require treatment as a primary condition or is it generally self-resolving when the underlying pancreatic problem is addressed?

RK Pancreatic ascites is a relatively dangerous condition because if pancreatic enzymes become active or are secreted in an active state outside the duodenum, they can cause potentially debilitating or fatal digestion of the tissues within the belly cavity.

G&H What has been the historic scenario for immediate treatment of ascites?

RK Prior to approximately 15 years ago, all cases of pancreatic ascites and high amylase pleural effusions were treated with total parenteral nutrition to minimize pancreatic secretion, administration of diuretics, and large-volume thoracentesis and paracentesis to remove the pancreatic juices. Octreotide or another somatostatin analog was also given to stop pancreatic secretion. If patients did not respond within 6 weeks on that therapy, salvage surgery was performed, either resecting part of the pancreas in the area of leakage, or, in the case of a concomitant pseudocyst, cyst-gastrostomy or Roux-en-Y cyst-jejunostomy.

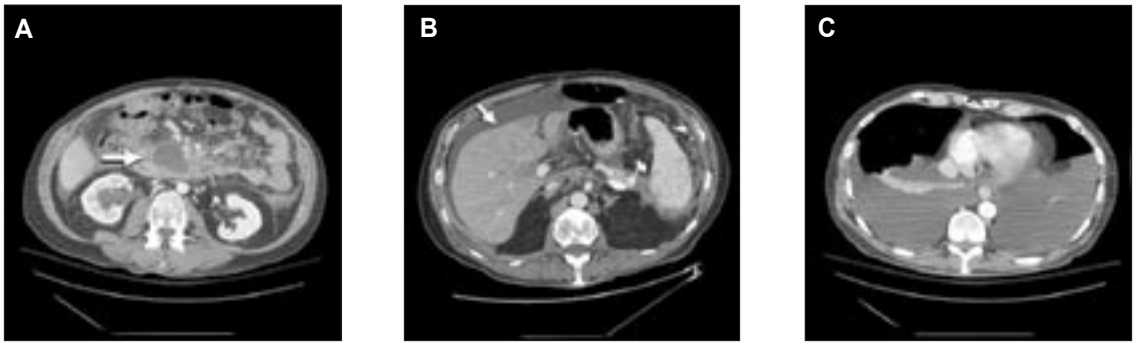

Figure 1. Abdominal computed tomography demonstrates retroperitoneal edema and pseudocyst (arrow) (A) in a patient with pancreatic ascites (arrow) (B) and bilateral high amylase pleural effusions (C).

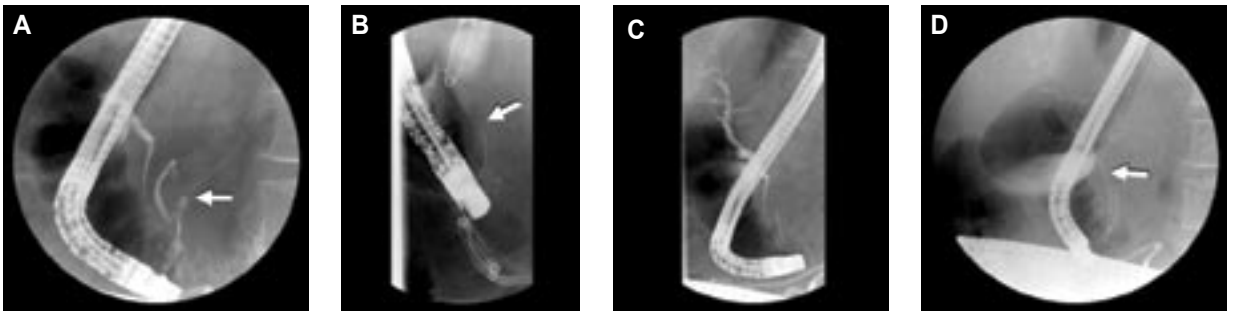

Figure 2. Endoscopic retrograde cholangiopancreatography demonstrates high-grade intrapancreatic distal common bile duct stricture and leak of pancreatic duct (PD; arrow) at genu (A). Arrows demonstrate guidewire insertion into percutaneously drained pseudocyst (B). Following transpapillary PD stent placement (C), the bile duct is selectively cannulated, and a biliary stent (arrow) inserted for concomitant cholestasis (D). This patient resolved his PD leak/cholestasis with combined endoscopic and interventional radiologic therapy.

This approach led to healing in less than 50% of patients and, among all patients, those who healed and those who subsequently required surgery, relatively high morbidity and mortality. With this method, there was an approximate 10–15% overall mortality rate and a 15–25% recurrence rate.

G&H How have endoscopic procedures allowed for more effective treatment of pancreatic ascites?

RK Our group initially described the process of performing endoscopic retrograde cholangiopancreatography (ERCP) to locate the site of disruption and, subsequently, placement of a transpapillary stent across the leak site to bypass the obstruction, in addition to large-volume paracentesis. If necessary, patients undergo concomitant endoscopic or percutaneous pseudocyst drainage. This puts the duodenum in a low-pressure system that allows the pancreas to decompress itself into the duodenum. This approach, for patients with amenable anatomy, has changed treatment of pancreatic ascites worldwide since we initially described it in the early 1990s. There are now a large number of published case series and meta-analyses showing that if the site of ductal disruption can be bridged, pancreatic secretions can be kept from leaking intra-abdominally, and the need for urgent surgery avoided in the vast majority of these patients. I have patients who underwent this procedure 15 years ago and have never had a recurrence. We have a very active team approach among our interventional radiologists, pancreatico-biliary surgeons, and therapeutic endoscopists, who work together to treat these patients.

G&H Has the endoscopic procedure changed over the years as endoscopic and imaging technologies have advanced?

RK The advent of helical computed tomography has allowed us to detect contiguous fluid collections and any upstream dilation behind a stricture or stone. Further, imaging with magnetic resonance cholangiopancreatography (MRCP) allows us to take a pancreatogram and give secretin to define the exact site of the disruption. MRCP can also be used to follow patients without the risks associated with repeat ERCP. In this way, 4 weeks after stent placement, the pancreas can be stimulated to confirm that juice drains into the duodenum and does not go out a continued ductal disruption.

Additionally, there has been a subset of patients with lesions that were not endoscopically amenable historically, because of a disconnected gland syndrome where there is a piece of pancreas at the head and one at the tail, as a consequence of central pancreatic necrosis. Currently, endoscopic ultrasound allows us to perform transgastric punctures into dilated pancreatic ducts that may be leaking and place transgastric or transduodenal stents to alleviate this leak. Thus, these patients, who always required surgery in the past for a disconnected gland and who usually had associated fluid collections to include pancreatic ascites, can often be managed with endoscopic ultrasound-directed therapy.

G&H Is endoscopic treatment now considered the gold standard in these patients?

RK These disorders should be treated endoscopically in tertiary referral centers. The old methods of 4–6 weeks of total parenteral nutrition and octreotide treatment are obsolete because if a duct is leaking into the peritoneal cavity, there is generally an ongoing structural lesion causing it. It is now understood that those leaks need to be addressed endoscopically or surgically both to initially treat and to prevent recurrence. These are very complex problems with very high rates of morbidity and mortality when they are not treated properly.

Currently, we are looking at leakage as a cause for, or consequence of, pancreatic necrosis in a significant subset of patients. Our hope is that stents placed early in the process of pancreatic necrosis will moderate the disease process and alter the outcomes of morbidity and mortality.

Suggested Reading

Kozarek RA. Therapeutic pancreatic endoscopy. *Endoscopy*. 2001;33:39-45.

Kozarek RA, Ball TJ, Patterson DJ, et al. Transpapillary stenting for pancreaticocutaneous fistulas. *J Gastrointest Surg*. 1997;1:357-361.

Kozarek RA, Jiranek GC, Traveerso LW. Endoscopic treatment of pancreatic ascites. *Am J Surg*. 1994;168:223-226.